

conserved. This suggests that they likely share co-factor binding specificity. In *Drosophila*, 25 proteins that can interact with SO have been identified (Giot et al., 2003; Kenyon et al., 2005). We performed database searches for putative *Xenopus* orthologues of these fly genes and identified between one and four clones with sequence homology to 15 of them. Expression assays show that 20 *Xenopus* orthologues of 10/15 of the fly genes are expressed in the developing placodes as well as other craniofacial tissues. These results suggest that there are novel co-factors that may regulate the ability of Six1 to promote and maintain cranial placodes during development. Functional assays will determine the roles of these proteins in placode development. (Supported by NIH HD055321 and NSF IOS-0817902).

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#### Program/Abstract # 135

##### **Chicken Scratch2 is expressed during early embryonic neurogenesis**

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In invertebrates, the *Scratch* (*Scrt*) genes encode transcription factors that promote neurogenesis during development. The *Scrt* function in vertebrates is currently unknown, but in mice *Scrt1* and *Scrt2* are specifically expressed in post-mitotic neurons in the embryo and in the adult central nervous system. In this work, we have cloned the coding sequence of chicken *Scrt2* (*cScrt2*) and characterized its expression pattern in the embryo with RT-PCR and *in situ* hybridization. The complete coding sequence was cloned and the predicted translation product is a 276-aminoacids protein. This aminoacid sequence shares identities of 70% with rat *Scrt2* and 58% with zebrafish *Scrt*. *cScrt2* transcripts are first detected as a faint signal in the periphery of the neural tube in the hindbrain by HH 15 and in the spinal cord by HH 17. The intensity of the signal increases between HH 19–23, and the expression in the motor domain of the spinal cord is progressively concentrated in the interface between the ventricular and mantle zones. *cScrt2* expression is also observed in the dorsal root ganglia after HH 22–23, particularly in the dorsomedial domain. The expression pattern of *cScrt2* in the neural tube is complementary to that of *Notch1*, which is expressed in neural stem cells, and to that of *SCG10*, a marker for differentiated neurons. Our results suggest that during embryogenesis *cScrt2* is expressed in a population of post-mitotic undifferentiated neurons.

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#### Program/Abstract # 136

##### **Concentration-dependence of Tcf3's function in Wnt/ $\beta$ -catenin signaling**

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Tcf/Lef proteins, the downstream transcription factors of Wnt/ $\beta$ -catenin signaling, can act as either transcriptional activators or repressors. Loss of function experiments in several organisms were all consistent with the first embryonic function of the Tcf3 protein being independent of  $\beta$ -catenin interaction. In order to identify any embryonic role for Tcf3/ $\beta$ -catenin interaction, Tcf3 $\Delta$ N knock-in mice were

generated. Mice homozygous for the Tcf3 $\Delta$ N knock-in mutation, in which Tcf3 cannot bind to  $\beta$ -catenin, exhibited several morphogenetic defects, including exencephaly, poor vascular integrity, open eyelids at birth and oligodactyly. Interestingly, all these defects were rescued by reducing Tcf3 protein levels in Tcf3-/ $\Delta$ N mice. Furthermore, Tcf3 overexpressed specifically in the eyelid epithelium of transgenic mice caused eye-open phenotype. These results, combined with BAT-GAL reporter gene activity, indicate that low levels of Tcf3 allowed Wnt/ $\beta$ -catenin signaling while high levels of Tcf3 inhibited Wnt/ $\beta$ -catenin signaling. Reducing nuclear Tcf protein to transduce the Wnt signaling was previously described for the "variant" Wnt pathway in *C. elegans* (Wnt/ $\beta$ -catenin asymmetry pathway). Taken together, these findings suggest this so called variant mechanism of Wnt/ $\beta$ -catenin activation of target genes, by stabilizing  $\beta$ -catenin and decreasing nuclear Tcf protein, may be more broadly conserved than previously appreciated.

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#### Program/Abstract # 137

##### **Deciphering the mechanism of Engrailed function during mouse cerebellar foliation**

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During mammalian development, the cerebellum (Cb) arises from rhombomere 1 of the developing hindbrain. The mammalian homologs of the fly segmentation gene *engrailed* (*en*), *En1* and *En2*, are required for specification of the cerebellar anlage and have been implicated in late patterning of the developing Cb. However, little is known about the mechanism(s) by which the *Engrailed* genes regulate later cerebellar patterning. Within the developing cerebellar primordium, two main regions of neurogenesis exist, the ventricular zone and rhombic lip, which give rise to Purkinje and granule cells, respectively. During late embryonic and early postnatal development, the Cb undergoes a drastic increase in tissue size accompanied by folding of the Cb along the A–P axis into lobules. The formation of these lobules occurs through the initiation of fissures and the proliferation of granule cells. Between E17–18.5, four primary fissures form and separate the Cb into five lobes. We show that leading up to and during the specification of these fissures, the *Engrailed* genes are expressed dynamically in cells derived from both the ventricular zone and rhombic lip. Using tissue-specific conditional gene inactivation, we show that *En1* and *En2* are required early for specification and/or production of cells derived from the ventricular zone. Furthermore, we show that loss of *En1* and *En2* in ventricular zone derived cells or in granule cells results in severe A–P patterning defects. We propose that the *Engrailed* genes pattern cerebellar foliation along the A–P axis by specifying positional and temporal cues, which result in formation of the four primary fissures.

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#### Program/Abstract # 138

##### **Characterization of the function of Sox21 during *Xenopus laevis* neural development**

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Neurogenesis, the progression from progenitor cell to committed neuron, is fundamental for the development of the central nervous system (CNS), yet the mechanisms involved in this process are not well defined. Members of the SoxB family of transcription factors have been shown to play important roles in neurogenesis. SoxB1 proteins are required for induction of the CNS and maintenance of a neural stem cell